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Acknowledged

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[Reprint from *The Laryngoscope*, St. Louis, October, 1904.]

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PURULENT OTITIS MEDIA COMPLICATING TYPHOID FEVER.

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That the ear complications of typhoid fever have never received the attention they deserve is shown by the paucity of literature on the subject. The reason, doubtless, is that it is almost never fatal in a few days, as is often the case with hemorrhage, pneumonia, perforation, etc., yet it is in some instances as grave as any of these, though not being so immediately serious, its true import is overlooked. The near or remote future, with its possibilities of mastoid empyema, intracranial complications, or chronic suppurative otitis, deafness, foul otorrhea, facial paralysis, etc., is lost sight of. Lermoyez states that the staff of a French hospital told him that the acute otitis cases, (in children's eruptive diseases) were very mild, a little carbolated glycerine being all they needed. At the same time they mentioned that most of the cases left the hospital with ears still discharging slightly. Comment is unnecessary before of otologists.

Deafness in typhoid fever. It is not contemplated in this paper to consider the non-suppurative ear complications further than they may bear, or might be thought to bear on the suppurative conditions. All clinicians have noticed lessened auditory acuity in typhoid fever. This has been attributed to blunted perception, deemed central and due to toxemia.

In an examination of the ears in typhoid fever, we were at once struck with the fact that practically all cases could be classified into two great classes, dullness of hearing with and without evidence of middle ear inflammation. This dullness of hearing was analyzed as to etiology in 51 selected non-suppurative cases. It was found to be due to the tube in one, because the patient, a mild case, volunteered the statement that he heard better after swallowing 3 or 4 times, and on test was found to hear normally. Only 12 cases were catheterized as a test and they were all patulous. Dullness of hearing was found to be due to non-suppurative middle ear conditions in 17 cases, (some of these might have been tubal). The perceptive apparatus, including the sensorium, was found at fault in 23 cases; both perceptive and conductive mechanisms in ten cases.

These data must not be taken as absolutely correct for very apparent reasons. The cases were selected for their mildness, their

intelligence and the speaking of a language of which we had at the time a skillful interpreter. They were all examined between the 10th and 20th days of their illness.

With a view of gaining a little information in regard to otitis media purulenta acuta, a complication of typhoid fever, a number of series of observations during a year past were made in the medical wards of the Western Pennsylvania Hospital at Pittsburg. This series of cases embraced first 280 cases of typhoid fever examined in the nose, throat and ear on admission and at intervals afterward. Other series embracing the purulent otitis cases of the first series and various other series were analyzed from carefully kept records. It was not possible to follow every case in all the lines of investigation. This accounts for the many separate series of cases, with different numbers in each.

In further considering the observations here recorded percentages, etc., it must be remembered that these were all hospital cases that they were consequently nearly all severe infections, aggravated before admission by working during the early stages, by the eating of a general diet, and by all kinds of neglect. No cases of paratyphoid fever are included, so far as known.

OTITIS EXTERNA.

Two cases of otitis externa circumsonpta occurred. This seems a rare complication. Bezold in 1243 cases of typhoid fever with 50 cases of ear involvement did not see a single case of otitis externa. No other writer mentions it. In one of our cases, free incision and drainage with a little wick of gauze resulted in a slow but uninterrupted recovery. In the other case, the process penetrated deeply, setting up a periostitis. An incision down to the bone extending from near the tympanic membrane out to the tragus, with occasional currettement, packing with gauze, etc., was the treatment; but in spite of this, the pus worked its way outward and developed quite a collection in front of the tragus. An incision here, with through drainage by means of a wick of gauze pushed in the external wound and brought out in the canal, brought about a slow recovery of 7 weeks duration. It was a continual fight with exuberant, flabby granulations and tissues reluctant to heal, on account of a system profoundly poisoned by toxines.

That external otitis did not occur often in the suppurative cases is remarkable, when the virulence of the pus is considered; and also when we consider the frequency with which the ear is fingered by the patient. As mentioned under bacteriology, the external canal was always found sterile at the deeper osseous portions, but

infected near the concha. The latter location is, of course, where furuncles usually develop, and was the location in the two cases mentioned. That it did not occur oftener, considering the frequency of median otitis, seems to point to a lessened resistance of the mucosa, and an undiminished resistance of the skin.

DISORDER OF THE PERCEPTIVE APPARATUS.

No special investigation of this was made further than to record its presence. Abnormality of the perceptive apparatus was found in 33 out of 55 typhoid fever cases. It came on in the first or second week and lasted until the temperature reached the normal.

Dr. Dench in his classical text-book, under the head of typhoid fever involvement of the perceptive apparatus, attributes the diminished sound perception to the effect of the specific poison on the cerebrum itself, rather than on the nerve distribution in the labyrinth, on account of the disappearance of the impairment of hearing during convalescense. This seems to be borne out in a few of our non-tympanic cases, but in the most of them, the annoyance produced by severe tinnitus seemed to indicate that the toxemia was acting more powerfully on the labyrinthine nerve endings than on the sensorium. It has been argued by Politzer and others that the earlier disturbances of the labyrinth are hyperemic and the later disturbances anemic. This seems refuted by the fact that there is not the hyperemia in any portion of the head in typhoid fever that there is in, for instance, pneumonia, which is free from non-suppurative labyrinthine trouble. As to anemia as a factor it is present during a considerable part of the convalescense, while the labyrinthine trouble ordinarily ceases on the eve of convalescense.

ACUTE CATARRHAL OTITIS MEDIA.

In 8 out of 280 (2.9 per cent) cases of typhoid fever there were evidences of a mild otitis which subsided without rupture or incision of the drum membrane. We did not find catarrhal otitis as common as the catarrhal affection of the upper air passages. Cases which went on to purulent otitis were not included in the figures given.

MYRINGITIS.

Six cases of this were found in 280 cases (2.1 per cent) of typhoid fever. Of course only those cases were counted that showed no middle ear affection.

ACUTE PURULENT OTITIS MEDIA.

Literature. The earliest and also the most erroneous reference to ear affection in typhoid (or typhus fever) is that of Frascator[1]

in 1564, who said *"surditas salutem portendit."* The same error was voiced three centuries later by Allison[2] who in 1849 said "Deafness is rather a favorable symptom in typhoid."

The earliest record found of otorrhea was that of Louis[3] who in 1841, refers to ear suppuration followed by meningitis. Peacock in 1856,[4] Wilson in 1881,[5] and Murchison[8] all report similar cases.

Murchison[8] writes: "I have known rigors, high fever, intense headache and delirium and even convulsions, occur during convalescense, and cease at once on the appearance of discharge from the ear."

Trosseau[6] in 1861, states that if both ears be affected, it is usually due to a catarrhal extension from naso-pharynx through the Eustachian tube; whereas, if but one ear be affected it is due to a purulent otitis, an occasion for graver prognosis.

Oppolzer[7] attributes deafness in typhoid fever to three causes; Catarrhal extension from naso-pharynx; central nervous disorder from blood dyscrasia; periostitis of the middle ear. Of these catarrhal extension, he regards as common, central nervous disturbance rare, periostitis he considered metastatic in origin.

Moos is quoted by Bezold as reporting two cases of total deafness which were referred back by the patients to an earlier attack of typhoid fever. One had bilateral deafness with residua of a purulent inflammation; both drum membranes being sunken in darkened and perforated in the center. In the other case, the drum cavities and drum heads seemed normal on both sides.

A degree of inaccuracy exists in the earlier observations, especially the European, in that typhoid and typhus (which also has ear complications) are in some instances considered together. Of course, in later observations this does not occur.

Coomby[9] considers deafness and tinnitus pathognomonic of typhoid fever. Nearly all the leading text books on internal medicine refer to deafness, a few of them to tinnitus, as symptoms in the second week of typhoid fever. This, of course, independent of suppuration. Few mention suppuration, and only two give any suggestion as to its treatment.

Frequency. The exact frequency of otitis media purulenta as a complication of typhoid fever is exceedingly difficult to determine. It might be expected to vary in the different epidemics, different countries and different social conditions and surroundings. Few statistics are available because, with one or two exceptions painstaking records have not been made.

Bezold in four years (1876-80) saw 1243 cases of typhoid fever and saw 48 (3.9%) ear complications exclusive of purely nerve con-

ditions and the dullness of hearing due to antipyretics (presumably quinine at that date). This is an astonishingly low percentage.

Louis records that out of 45 severe cases, of typhoid fever that recovered, the ear in four cases (8.9%) had discharged, and though a perforation was observed in one case only, it is probable that all were median otitis. In 24 mild cases, one (4.2%) had suppurative ear trouble, though three complained of ear pain. In 30 cases that died of typhoid fever he noted no case of suppuration—probably, we suggest, because they died too early in the disease.

Beverly Robertson[10] states that "otorrhea" is more frequent among children than with adults, but does not give his observations of the frequency of either.

Solis Cohen and Claribel Cone in 1893, report a case of purulent otitis "deemed worthy of record because it was of rare occurrence in this hospital" (Philadelphia) although it healed with a perforation, but without otorrhea or mastoid or other complications.

Raoult and Specker[12] give no figures, but make the general statement that otitis media is a very frequent complication of typhoid fever. This is in 1902 and probably refers to cases occurring in Nancy, France.

Hengst in 1896, collected 28 cases of purulent median otitis occurring in 1228 cases of typhoid fever. Five hundred and seventy-five of these cases were private cases. The percentage, 2½, was about the same in both classes of patients.

Stricker reports 34 cases of otitis out of 172 cases of typhoid fever, (19.7%). Jessen, 13 out of 187 (7%). Hoffman, 4 out of 250 autopsies (1.6%). Osler, 8 out of 389 (2.1%).

Vickery reports for four months, July to November, in 1902, at the Massachusetts General Hospital, in 49 cases of typhoid fever, two instances of purulent otitis (4%) one of pneumonia and three of hemorrhage.

For 1902, in the Boston Hospital, Sears reports 11 cases of otitis media in 203 (5.4%) typhoid fever cases, standing next to pneumonia (12 cases) and phlebitis (12 cases) in frequency as a complication. In comparing such reports with the percentages given in our cases it must be borne in mind that in few if any of the hospitals can the records of the ear complications of typhoid fever be relied upon for completeness. We made almost daily visits to the hospital and made it a duty to record all cases of otitis during an entire year.

Systematic inquiry into the frequency of purulent otitis in typhoid fever in private practice has not yet been made by us, as it seemed,

in making personal inquiries, that mental records would be the only ones available, and these are notoriously unreliable.

Personal inquiry among general practitioners of large practice, revealed that eight of them never saw a case among several hundred typhoid fever cases. These men said they rarely saw a severe case of typhoid fever in private practice. A mild type was the rule. Four men in large practice in the poorer districts frequently saw very severe and fatal cases of typhoid fever and occasionally saw a discharging ear. This seems to point to the hereinafter mentioned observation that the severity of the case is an etiologic factor.

Coming to our own observations in the Western Pennsylvania Hospital at Pittsburg, in 780 cases of typhoid fever, otitis media purulenta acuta was noted in 88 cases (11.3%) 45 times (51.1%) it was only on one side (26 left, 19 right); 43 times (48.9%) on both sides. It was during this period the most frequent of all the complications. It has developed this prevalence in recent years, though it is greatly on the decrease in the last few months and at the present time it is down to one case in 64 (1.6%). Statistics on the frequency in this hospital prior to 1903 are not available, so that the very interesting point as to whether or not these cases occur more frequently in recent years cannot be proven in figures.

Statistics on this point are difficult to obtain and are valueless. There has never been in most hospitals, any attempt at recording these complications conscientiously. It certainly does not appear at all in many reports and in others it has only appeared in recent years. Former residents and nurses in the Western Pennsylvania Hospital remember occasional cases, but all are certain that it was less frequent formerly.

Those practitioners in private practice who have had cases were inclined to look upon it as a complication of recent prevalence. Mention is made of it in literature many years back, yet in Pittsburg, at least, it is certainly more prevalent now than ever before and it is certainly more frequent in the hospitals than in private practice.

One thing is quite certain, purulent otitis media is never overlooked when present in typhoid fever. It is accompanied by such a copious outpouring of sanguinolent serum in the early stages that it could never escape observation.

BACTERIOLOGY.

Acute otitis media independent of typhoid fever is usually but not invariably, monomicrobic. Chronic purulent otitis media is invariably polymicrobic; usually staphyloccocal with saprophytic mixture. Theoretically it would seem unlikely that there is a true typhoid in-

flammation of the membranous lining of the middle ear as in diphtheria, etc., on account of the difference in the selective properties of the organisms. Eberths bacilli do not attack the naso-pharyngeal mucosa as Klebs-Loeffler bacilli do. Eberths bacillus is pyogenic as proven by Mya, Belfanti, Dmochowsky and Janowsky, Black, Orloff and Gilbert and Girode. It has been found more or less abundantly in many of the purulent complications of typhoid fever.

Destree, Prochaska and Preysing have found it in suppurating ears of typhoid fever patients. Coplin found it associated with pyogenic cocci in a case of mastoiditis in typhoid patients. Many observers have not found it. Possibly it may have been present earlier and disappeared after the later mixed infections. Funke reports two cases of typhoid bacillus present in middle ear suppuration in pure culture and two cases associated with pyogenic cocci. He reported the organisms most common in the pus of otitis media in order of frequency as follows:

Pneumococcus,
Streptococcus,
Pyogenic Staphylococci (Albus and Aureus),
Bacillus of Friedländer,
Bacillus diptheriæ.

Durin in two bacteriological examinations did not find typhoid bacilli in purulent otitis complicating typhoid fever The diplococcus of pneumonia has been found by several observers. Fraenkel and Simmonds found pyogenic bacteria in middle ear discharges but were unable to find the typhoid bacillus.

A review of the literature will indicate clearly that we are not aware of all the factors involved in the sterility or infection of the middle ear. Thus Zaufel, from investigation on rabbits, concluded the tympanum is by no means always sterile, the number of bacteria increasing the nearer the approach to the pharyngeal orifice of the tube. On the other hand, Preysing out of 78 ears found (post-mortem) 67 sterile and 11 infected, but apparently healthy, from which he concluded that the normal ear is practically sterile. We are somewhat in the dark as to the number of bacteria required to start an infective disease, not in the ear only but in any tissue. Doubtless the number varies widely dependent upon many factors. In the normal ear as in other organs it is reasonably certain that a limited number of pathogenic organisms may be and often are present without lesion. Exactly the proportionate share in the protection taken by the body juices, by healthy epethelium, adequate drainage and phagocytosis or its equivalent in cell action, is not agreed

upon. It is, however, an accepted fact that there is a protective power exerted by the healthy organism, which power varies greatly and becomes very much lowered in a great variety of acute and chronic ailments. It is possible also that these ailments increase the virulence of the bacteria so that their attacking power is greater; for it is now well known that pathogenic power is not necessarily in proportion to numbers, virulence being equally a factor.

Bacillus diphtheriæ has been found (Prochaska) associated with pus cocci in otitis pus in typhoid fever cases, although undemonstrated in the throat. The bacillus typhosis has been found in typhoid fever in surgical complications of the bones, periosteum and cartilages, in abscesses of ovary, spleen, liver, kidney and cellular tissue; in orchitis, epididymitis, thyroiditis, localized peritonitis, endocarditis, cholecystitis, meningitis, pleurisy and a number of other conditions and locations. In all these there was suppuration, but the bacillus typhosis has been found without lesion in bone marrow and in lymphatic glands.

All the mucous lined cavities of the body have been shown to intermittently, if not constantly, contain a varying number of pathogenic organisms, which owing to defensive power of normal epithelium, ciliated or not, of body juices and of leucocytes, gain no foothold. It is a well recognized fact that in typhoid fever we have a very low resisting power as evidenced by the long list of pyogenic invasions of nearly every organ in the body by organisms every day carried about with immunity by the normal individual.

How far the virulence of bacillus typhosis might be increased by being confined in a closed cavity like the middle ear, where the contents are not constantly moving, can only be conjectured.

Coming to the results of our investigations, with the assistance of Dr. Ralph Duffy, pathologist of Western Pennsylvania Hospital cultures were taken from the naso-pharynges and external auditory canals of 60 cases of typhoid fever, on admission. The canal close to the drum membrane was in all cases found free from pyogenic organisms.

In four cases of purulent otitis media a culture taken two, three, six and ten days, respectively, before the onset of the otitis media showed the deeper canal sterile. While this was an abundantly long time to permit deeper penetration, yet taken in conjunction with the fact that no case at any stage was found with an infected canal (in its deeper portions) we think argues against the theory of infection by this route.

The bacteriological examinations of the pus from the middle ear in 22 cases showed staphylococcus aureus in pure culture four times

streptococcus in pure culture eight times and these two in mixed infections six times. The pneumococcus was found twice. Bacillus diphtheriæ twice.

In seven of the eight cases where streptococcus was found as a pure culture in the pus from the ear, the same organism was found in the naso-pharynx. Staphylococcus was demonstrated in the naso-pharynges of all of the ten cases in which it was present in pure culture in the ear pus.

The typhoid bacillus was not found in any case in either the naso-pharynx or the ear discharges. Theoretically it seemed plausible that the bacillus typhosis might be present as an initial factor, mixed infections with pus cocci developing later. With the object of determining this, eight cases were incised early and the cultures taken with a sterile probe inserted through the incision, before there had been opportunity for the proliferation of air borne organisms. In no case was the typhoid bacillus found, though pus cocci always were, evidently having gained entrance through the Eustachian tube. Evidently in both classes of cases (the one we believe due primarily to infection, the other to embolism or thrombosis) the pyogenic organisms were evidently present but were unable to proliferate freely until the required conditions of soil were developed. To prove this, in examining the external auditory canals of typhoid fever cases, free from otitis, in three instances the membrane was incised under aseptic precautions and a culture taken from the mucosa of the middle ear itself. Again no typhoid bacilli were found, but in all staphylococci were found. In all three of these cases, this examination was made in the second week of the disease. All of them got well of their fever, without developing otitis. Presumably this was because of lack of conditions of soil, be those conditions what they may—an inflammation of the mucosa due to typhoid bacilli lodged from the blood flowing through the mucosal vessels; or a low resistance of cells or juices due to toxemia; or to a necrosis of the mucosa from anemia due to embolism or thrombosis, or to an anemia from any other cause. Or the typhoid bacillus by causing a non-inflammatory tissue necrosis may have prepared the soil for other more active pyogenic bacteria. As might be inferred from the statement just made, it is not intended to argue from these examinations that the typhoid bacilli may not be the chief etiologic factor because it was not found in the middle ear or in the discharges therefrom. Its attack may have been from beneath the surface by way of the mucosal vessels, and may have even there been rapidly superceded by the all pervading, ever present, pus cocci.

The presence of colon bacilli was suspected to be present in one case on account of the fecal odor of the pus, but it was not found.

The naso-pharynges of 60 typhoid fever cases were all found free from infection with typhoid bacilli. This is remarkable when it is remembered that the sputum almost always contains these organisms. It may be mentioned here as a control, that the same laboratory technique was demonstrating the presence of typhoid bacilli in suppurative complications, other than those of the ear. The pathogenic organism found in the naso-pharynx were:

Streptococcus,
Staphylococcus Aureus,
Staphylococcus Albus.
Pneumococcus,
Bacillus Diphtheriæ.

Besides these there were numerous saprophytes and air organisms.

Before leaving the subject of bacteriology it might be well to recall to our minds the fact that it is now conceded that glandular structures may be the seat of inflammation in the absence of bacteria. Whether the glandular apparatus of the tympanic mucosa can be the seat of such an inflammation we are unable to say.

PATHOLOGY.

It is to be regretted that the difference between the slow and the fulminating types was not noted earlier in the investigations. Had this been done, our observations would have all been with a view to throwing light on the differences in the pathology of the types. It is also to be regretted that there was no opportunity for post mortem investigations into the pathology and pathological histology. It is to be hoped that both these defects will be overcome by a future series of observations.

Bezold considered it open to question whether some of the cases of post typhoid deafness were due to extension to the labyrinth from the middle ear with destruction of the fenestra; or whether it were not more likely that an independent suppurating focus developed in the labyrinth similar to that in cerebro-spinal meningitis. He admits that no such case has been observed. We do not see any basis for it, for all the evidence we have indicates that the labyrinthine symptoms present in non-suppurative typhoid fever ear cases are due, not to infection, but to the circulation of toxines in the labyrinthine vessels.

In cerebro-spinal meningitis we have an infective focus extending outward from within the cranial cavity. No such condition exists

in typhoid fever. The infective focus is in the middle ear and has been observed by the writers to involve the inner tympanic wall in two cases. In both these cases, there were labyrinthine complications with permanent total deafness, (fortunately unilateral) and temporary vertigo. There was no facial paralysis.

It is unnecessary before this learned body to review the pathology of acute purulent otitis media, as you are familiar with the stages of vascular engorgement followed closely by transudation of blood fluids, the migration of white blood cells and the pouring out of catarrhal secretions, etc. What we have classed as the slow type of cases seemed in the absence of autopsies to be pathologically the same as that of acute otitis media, occurring independent of typhoid fever, except that it was a very intense grade of inflammation. The two types of cases, the slow and the fulminating after the end of about a week or ten days assumed the same pathological state so far as the gross pathology could be conjectured from the naked eye appearances. The difference was in the rapidity of onset. The fulminating cases reached in a day, the stage that the cases of slow type reached in four or six days. The naked eye appearance which is all the source of information as to pathology that we have to offer was partially described under "symptomatology." Some further observations follow: The mucosa of the tympanum as seen through incisions and perforations in the membrana tympani, was, in the slow cases, sometimes deep red in the later stages, always so in the early stages. Frequently in ten days or two weeks the membrane would become pale and edematous, as though covered with flabby granulations which were pouring out pus. In many of all three classes of cases, large masses of pseudo-membranous exudate had to be wiped away from the fundus of the canal in the early days of the suppuration. It did not leave a bleeding surface. Microscopical examination of this substance showed it to be exfoliated epithelium, with, in some instances a fribrinous net work. In six cases of suppurative otitis, on the second day the membrana tympani was incised all round the tympanic ring. After the bleeding had been stopped with dry cotton tampons the edge of the membrane was turned up with a probe in successive portions and a careful examination of the atrial mucosa made. In five other cases the membrana tympani was cut in quadrant flaps by a vertical incision from top to bottom of the tympanic ring anterior to the malleus; a horizontal incision crossing this from anterior to posterior periphery just below the umbo; and a third incision vertically close to the malleus posteriorly. These two plans of incision permitted, after hemorrhage stopped, a free inspection

of the atrial mucosa. In all these cases the membrane was bulging with the presence of fluid discharges. Inspection showed three different states of the mucosa.

(a) Intensely reddened and inflamed with slight swelling associated with sero-sanguinolent discharge.

(b) The same with gray sloughing patches of exudate looking in spots as if touched with silver nitrate.

(c) Intensely swollen, rather pale œdematous mucosa that obliterated all landmarks. The fundus of the canal looking as if filled with exuberant granulations.

These eleven cases were not examined until after the appearance of the discharge. They had not complained of pain and they occurred during a period when no typhoid cases were being examined aurally, unless ear symptoms were noted by the nurses or internes, so that it could not be stated to which type they belonged. Furthermore, they occurred before it was realized that there were two types of cases in addition to the hemorrhagic. In one very acute case of the fulminating type, there seemed to be an acute osteitis. The malleus and incus seemed to become necrotic and their attachments sloughed away. They both were wiped out on a cotton applicator. In this case the walls became slightly involved along the lower outer attic wall, where a small sequestrum came away, leaving a notch. This case recovered without operation apparently because of the ample drainage afforded.

In four cases of purpuric typhoid fever, in which the skin surface was mottled in various locations with dark blue spots, the mucosa of the pharynx, naso-pharynx, and larynx were seen to be spotted in the same way. The Eustachian eminence were free from the spots in all four cases. The mucosa of the ear in all four cases was examined as well as possible through tympanic perforations and was seen to be spotted with blue black discolorations in one case. In one other case the entire middle ear as far as could be seen was slate colored blending to brownish black and black anteriorly. This case did not bleed from the ear. It did bleed copiously from the nose. Another case bled freely from the ear. No exact source of hemorrhage could be located. The blood seemed to ooze through the blackened membrane everywhere. The blood was brown in color and oozed through a tight gauze plug like water. There seemed to be changes in the blood which deprived it of its coagulability. The hemorrhage was

only stopped by a cotton plug dipped in collodion forced tightly into place immediately after removing a drying plug of gauze. This corked the canal like a cork in a bottle. The pathology of the hemorrhage cases is not clear. Evidently there was a leaking out of blood through the wall of the mucosal vessels and through the epithelium into the cavity of the middle ear, which blood afforded an excellent culture medium for the proliferation of pus cocci. Two such cases were seen in their hemorrhagic stage, and watched until pus formed before evacuation by incision. No case of hemorrhage with subsequent absorption was seen. In another class of hemorrhagic cases of which five were seen, the hemorrhage was evidently under the epithelium. In two cases the blebs were incised, in three they were allowed to burst. The result was the same—an acute suppurative otitis media followed in all the hemorrhagic cases of whatever form. If it is assumed that the pathology of the blebs of the non-purpuric cases was the same as in the purpuric cases, the pathology is easily understood. But there is no evidence of this. The blebs were like little vesicles filled with a thin sero-sanguinolent fluid. When occurring on the drum membrane evidently the accummulation was between the layers. When the bleb was continued outward on to the canal wall, the surface of the bleb did not show any line of demarcation between what had been membranic epithelium and what had been parietal epithelium. The contents of the blebs was always apparently the same—fluid about the color of venous blood or slightly darker, but thinner and with no tendency to coagulate. The blebs were not accumulations in the anterior or posterior or Van Trœltsch's pockets. In one case of post suppurative sclerosis, many adhesions extending in every direction were found by the probe introduced through a rather small perforation, located just posterior to the umbo. All discharge had ceased seven years before after having continued two years following an attack of la grippe. This case was closely watched with special interest on account of the difficulty of the maintenance of drainage from the attic and aditus, should a suppurative inflammation occur. Two attempts at culture proved the middle ear sterile at the time the patient was admitted in the second week of his typhoid fever. Two weeks later he developed acute inflammation of the formerly diseased ear. The discharge escaped through the old perforation as soon as formed and never seemed to involve the attic. Of three other cases all developed a recrudescence of suppuration. One developed mastoid empyema, in which on opening it no sign of Sclerosing osteitis was found though there was a history of years chronic otitis.

The permanent impairment of hearing with lowering of the upper tone limit, in ten cases was doubtless due to small celled infiltration into some of the labyrinthine tissues. Whether this labyrinthine involvement was primary, due to irritation by the toxines in the blood circulating through the parts, or whether it was by extension from the middle ear, it is impossible to say as to all the cases. In three cases, however, this was a primary (not an extensionary) lesion in the labyrinth, for the middle ear was not affected. In four cases it was certainly an extension from the middle ear for there was necrosis of the inner (petrous) wall. Of course, in the latter cases it was impossible to say that there was no labyrinthine inflammation prior to the pyogenic extension. But the hearing had not been much affected until the onset of the median otitis.

In three cases, exuberant granulation pushed the drum head until it bulged as though retained secretions were behind it. Deeming the small perforation an insufficient outlet, a free incision was made, when instead of pus, a granulating mass pushed through like we see sometimes in chronic otitis media. This occurred after four, five and eight weeks, respectively, of otitis and was associated with dead bone in each instance.

Two cases when discharged had necrosis of the long process of the malleus, which refused to heal, and an operation was not permitted. Bone necrosis when occurring in the ossicles as it did in four cases was due, doubtless, more to interference with blood supply from pressure from the swollen mucosa than to simple virulence of the infection. It appeared in the walls in six cases, in three of which the process was constantly under observation. In these three cases the necrosis resulted after three, five and seven weeks respectively, of otitis. It resulted from the gradual deepening of an erosion that started as a superficial slough of the mucosa. When the bone was reached, the surface of the erosion was covered with a granular looking coating that looked not unlike mucous membrane, but through which the denuded bone could be felt with the probe. So far as direct observation was possible, the process always started superficially in the mucosa and not as an osteitis, though the exquisite tenderness in the early stages may possibly have been an osteitis. In one case the usual resisting power of the connective tissue at the vault was unavailing to ward off the streptococcal invasion and extensive necrosis followed. In this case when the mastoid was opened a large portion of the tegmen tympani was absent, but there was only a slight pachymeningitis which soon subsided.

ETIOLOGY.

Various theories backed by observations are advanced in the literature.

Demeurisse, who believed the otitis of la grippe and the acute infections contagious, thought the contagion occurred by the route "naso-tubaire."

Bezold classifies the causes as follows: Direct extension of naso-pharyngeal inflammation by continuity of tissue through the Eustachian tube; entrance of septic secretions into the middle ear through the tube; embolism in the mucous membrane from endocarditis or peripheral pyogenic-thrombi.

Politzer accepts Bezold's classification of causes.

Keen says it is "obviously a pyogenic infection from the throat." Hengst attributes it in part, to exposure of the side of the head to draughts; carelessness in washing and bathing; allowing the water to pass into the nose or into the external ears.

Hoffman believes otitis media purulenta in typhoid fever to be an extension from the naso-pharynx. Von Trœltsch and Blau consider dryness of the mouth and throat by closing the tubes with thickened dried mucus the chief factor.

Schwartz believes the trouble entirely independent of the tubes and to be a primary inflammation of the tympanic mucosa. Where tubal closure exists, he believes it is due to swelling at the tympanic end of the tube. Hartman considers the tympanic inflammation a specific typhoid fever inflammation similar to that in the intestines. This view is hardly tenable in view of the facts, first, that there is no lymphoid tissue in the tympanic cavity at all similar to Peyers patches, and second, the frequent onset of the ear inflammation is at a time when the specific enteric inflammation is subsiding. Of course, re-infections of the general malady occur at this stage and the ear inflammation might be regarded in this light as a late infection.

The following observations were made by the writers:

Among 41 cases of typhoid fever in private rooms of the Western Pennsylvania Hospital, one case, only, of otitis media (2.4%) developed. In the surgical wards but two cases developed in the house; one patient was of very low vitality because of anemia in the last stages of chronic nephritis, and the other seemed to have been infected from the naso-pharynx during anesthesia for an abdominal operation.

In the only case of otitis media purulenta that developed in the medical wards during the year, in a disease other than typhoid fever, was in a case of pneumonia and was due to the pneumo-coccus.

All these observations point to a predisposing, if not an exciting factor in typhoid fever that does not exist in other ailments. This factor, whatever it may be, is not usually present in the first three weeks. Bearing on this point, 93% of the purulent otitis media cases occurred after the 21st day; 8% between the 12th and 21st days. Of 20 cases of typhoid fever that died before the 21st day, no ear suppuration was noted. Of 20 cases that died between the 21st and 31st day of the disease, two had suppurating ears.

Naturally the causes are classified into predisposing and exciting factors. Whatever these may be, they never both exist prior to the 10th day of the general infection (not counting the prodromal period) and very rarely before the third week. This must certainly point strongly to the predominant etiologic factor.

What are the characteristics of the third week and the following weeks of typhoid fever as compared with the earlier period? Sensitiveness to cold, a rapid, often sudden emaciation, lowest resistance of the tissues and juices, thrombotic phenomena, glandular disturbance, pneumonia and broncho-pneumonia beginning inflammatory and purulent troubles, separation of the sloughs from the enteric lesions. The worst of the high temperature is over, but its damages remain.

In looking over the field of etiologic possibilities, a long list suggests itself.

Predisposing Factors.

1. Dorsal decubitus—a factor in drainage.
2. Swallowing, semi-somnolent and in a recumbent position, frequent and prolonged because of liquid diet. Repeated efforts because of difficulty in swallowing.
3. Acute catarrhal discharges in nose and naso-pharynx and ear in all typhoid fever cases. A good culture medium and an excitement of efforts at clearing by blowing the nose and hawking.
4. Lowered resistance, and weaker phagocytosis (or its equivalent)
   (a) Lessened tubal protective activity, weakened cilia,
   (b) Weakened resistance of tympanic tissues.
5. Increased virulence of organisms proliferating in the tympanic incubator.
6. Tubal closure.

7. Abnormal patency of the Eustachian tube.
   (a) Present before illness,
   (b) Due to absorption as a part of the general emaciation,
   (c) Due to ulceration and sloughing of tubal mucosa,
   (d) Due to violent activity of the levator palati mollis (temporary.)
8. Intensity of the general infection.
   (a) Pyrexia,
9. (b) Toxemia.
10. Perforated drum head from previous suppuration.
   (a) Admitting infection,
   (b) Facilitating infection per tubam.
11. Pretyphoid chronic catarrhal and purulent otitis.
12. Anemia of vascular tissue of nose, resulting in lessened filtration of inspired air, thus favoring infection of the naso-pharyngeal secretions.
13. Draughts of air especially when patient is perspiring, producing acute catarrhal otitis media, later becoming purulent by infection.
14. Hydrotherapy,
   (a) sponging
   (b) tubbing
   (c) wet pack
   (d) ice pack
   (e) ice bag on head.
15. Drugs.
16. Age.
17. Sex.
18. Nationality.
19. Occupation.
20. Impacted cerumen.
21. Associated typhoid and pre-typhoid disease of nose and throat.

Exciting Factors.

1. Extension by continuity of tissue.
2. Infection via
   (a) blood, infective emboli or typhoid bacilli floating free.
   (b) Eustachian tube
   (c) External auditory canal
   (d) Petro squamosal fissure
   (e) Lymphatics (Zaufel's lymph plexus).
3. Thrombosis
   (a) Aseptic clot
   (b) Infective emboli (classified under infection).

4. Sloughing of purpuric areas.
5. Erysipelas.

Let us take up these possibilities more in detail. Number one needs no comment further than to note that of 20 severe typhoid fever cases watched, they averaged 19 hours out of the 24 on their backs.

That swallowing facilitates infection by way of the tube seems likely. Swallowing is often difficult with typhoid fever cases, and the violent efforts bring into great activity the tubal dilating muscle, the tensor palati mollis. (spheno-salingo, staphylimes.)

The frequent, almost usual presence of a catarrhal state of the mucosa of the upper air passages in typhoid fever, is well known and was confirmed in these observations. But not so the ear. Out of 280 patients examined, 221 (78.9%) had catarrhal conditions of the upper air passages, while 21 out of 220 pairs (9.5%) of non-suppurative ears had evidence of a catarrhal state of the tympanic mucosa. From this, it appears that while the mucosa in the naso-pharynx may afford a culture medium for infective pus organisms, the tympanic secretions do not.

In regard to ærial infection of the naso-pharynx, the following observations are worthy of note:

C ward in which most of the cases here reported, occurred, is the oldest ward in the hospital. It is overcrowded practically all the time. On account of the crowded condition of the entire hospital it is not possible to remove the patients frequently for fumigation of the ward. Under these conditions it is not surprising to find in its air more pus organisms than in any other ward in the house.

In plate cultures made by exposure to the air of the ward for 1 hour and 40 minutes, large numbers of pyogenic organisms were found in the air. Approximately the same numbers were found in the medical wards of the other hospitals of the same age. The new hospitals showed more favorably. But even in these there were too many pyogenic organisms.

The accumulation of infective muco-pus in the naso-pharynx beause of the patient's feeble condition, being too weak to execute the necessary hawking, was a factor favoring infection via tubæ.

That efforts at blowing the nose and hawking are factors is doubtful, for purulent otitis media occurs almost exclusively in the severe typhoid fever infections, and these do not blow their nose, or hawk. The toxemia is too great. In fact it would seem that the lessening of the impulse to hawking and of the reflex coughing permits accumulation of mucus in naso-pharynx. The mucus

may be purulent from local inflammation or may be infective by reason of pus organisms collected from the inspired air. A longer sojourn than in the normally active naso-pharynx permits of incubation until a virulent, often a pure culture fills the naso-pharynx ready for aural invasion.

As to weaker ciliary activity it is a probability not proven.

The lessened resistance of the tissues in typhoid fever was demonstrated by Brieger and Ehrlich. Malignant edema followed the injection of a solution of musk in typhoid fever patients, but was harmless in others.

Liebermeister, years ago, wrote, "If one studies closely the manifold complications and sequelæ of typhoid fever, in the living and at autopsies, he will receive the impression that in severe cases the resistance is reduced to the minimum in all organs without exception, and that there is an extraordinary tendency to the destruction of the tissue." This expresses the clinical aspect of the predisposing factors. It is a condition probably brought about by the action of the toxines on the tissue cells and juices. This fact was not proven. It is given tentatively for future observations to prove.

(6 and 7). Tubal closure with its attendant congestion of tissue and exudation might, *a priori,* be deemed a factor. The condition of the tube, however, could only be determined positively by catheter inflation, and this did not seem a justifiable procedure in a disease with so many middle ear infections probably occuring per tubam. Nevertheless, the tube was tested and found patulous in all of 12 cases, more so than normal in ten of them. It was seen to be patulous in 6 suppurative cases by the pus oozing from the tube mouth in the naso-pharynx.

Ulceration of the mucosa of the tube mouth was found in four cases, all of which were followed, (not preceded) by purulent otitis. All unilateral. All started with a superficial gray slough, as if brushed with dilute silver nitrate solution. This deepened to a swollen ulcer with raised edges. In all three cases otitis developed within a week (third, fifth and seventh days respectively.) It ran a mild course and did not have the fulminating character seen in the cases believed to be due to embolism or thrombosis. In two of the cases, detached patches of the same gray slough were seen on the tympanic mucosa. They were detached and their entire circumference could be seen, so that they could not have been continuous with the tube mouth patches. The latter evidently arose from true typhoid infection in the glandular structure of the tube mouth (tonsil of Gerlach). It proceeded in one case so deeply that a

perichondritis tube resulted. The entire eminence became swollen to the size of a walnut, its mucosa highly edematous. Cartilaginous necrosis followed and part of the eminence sloughed off, leaving finally a pinhole tube orifice, with only a slight wrinkled elevation where the tubal eminence had been. This case had a mastoid empyema complicating the otitis on the same side as the tubal trouble. The start toward recovery of the tubal condition set in immediately after the mastoidectomy.

8. The severity of the typhoid fever infection is undoubtedly the most important factor in the production of purulent otitis.

Under the head of frequency, it was mentioned that inquiry among physicians in general practice developed the fact that those practitioners in the poorer districts occasionally saw cases of purulent otitis media, while those practicing among the wealthy class never saw even one case. The reason for severe cases in the poorer districts seems to be the continuance at labor in the earlier stages (some men working with a temperature of 103) and at the same time eating a general diet, often forcing food down in spite of anorexia, in the ignorant hope of keeping up the strength. Many laborers will make an untreated walking case out of conditions that would put the wealthier man in bed with bi-daily visits of the physician, and the constant attention of two trained nurses to enforce dietary and other restrictions and a proper regimen.

In our own cases it was noticed that out of 88 cases of purulent otitis, 71 (80.7%) occurred in cases in which the temperature exceeded 104.2 every day for a week or more, and in 30 (34.1%) the temperature reached 105. All other symptoms in a fair proportion of the cases kept pace with the temperature as indications of the severity of the case. No case of otitis occurred in any case in which the temperature did not exceed 102. In considering the temperature it must be remembered that except in cases not thought to require it the patient was sponged every time the temperature rose above 103. Wet packs and ice packs were used, if sponging failed to bring the temperature down.

10. That a perforation might admit infection seems plausible, yet our observations showing the external canal to be sterile would indicate that for infection to take place either infected fluids or strong currents of air would have to find their way to the fundus of the canal.

The previous aural history was usually unobtainable and was always unreliable when negative. Unless bilateral, ear disease among this class of people is usually disregarded and forgotten. Inspection, however, yielded accurate results in many cases.

A pre-typhoid chronic catarrhal otitis including sclerosis was observed in 21 cases, (7.5%). Of these one developed a purulent otitis in his normal ear, and one other case developed it in his catarrhal ear. This is of course negative, as in that month 10% of all the typhoid fever cases were having suppurating ears.

A chronic purulent otitis existing at the onset of the typhoid fever was always followed by an acute exacerbation, and this exacerbation, like the cases of primary acute otitis, occurred not earlier than the 10th day, and in three cases, not until the third week. The cases with pre-typhoid chronic purulent otitis differed in character from the primary cases. They discharged less and the discharge contained no blood or serum. They ran a milder course and did not seem prone to develop mastoid abcess. This milder character might bear on the etiology in various ways. Were the bacteria present, or was the granulation tissue resistent to a new infection of the type seen in the acute cases? Or does it only argue the existence of a predisposition to pus formation resulting in an acceleration of the pyogenic process already present? If this be so, does it not seem to argue that the other cases were embolic or thrombotic, while these few cases of chronic otitis escaped thrombosis?

(12) Anemia of the vascular tissue of the nose widening the air channel was present in practically all of the severe cases and seemed a part of the general emaciation. It showed most markedly in the third week and afterward; the period when most cases of otitis develop.

(13) The efficiency of a draught of air in the production of otitis would seem questionable if it were not for the constant presence of moisture on the skin. Just what way the chilling of the surface from the wind blowing on this moist surface might differ physiologically from the chilling of an ice pack, the writers are not prepared to say. However, we note that the wet pack or ice pack is used during pyrexia, while the most frequent occurrence of purulent otitis is later when the temperature has reached normal.

(14) It is Dr. John W. Boyce's practice to omit sponging in certain cases, no matter what the temperature.

He also does not advise the ice pack. He stimulates and feeds sparingly. This affords an opportunity for observing the effects of hydrotherapy including ice packs. Other members of the staff feed and stimulate more freely, the stimulants being chiefly strychnine and whiskey, and some of them use ice packs freely. Various drugs such as acetozone and guiacol were tried extensively by Dr. Lawrence Litchfield. Some patients liked an ice bag on their heads and were allowed to have it constantly, while others objected to it and it was not used.

Detailed tables would be too voluminous, but the result of our observations of all these differences in treatment and management lead us to conclude that hydrotherapy, including ice pack, and head ice bags (tubbing not being used) drugs, including stimulants, and foods and feeding are absolutely negative as etiologic factors in purulent otitis. Quinine has been considered a factor in the etiology of purulent otitis in typhoid fever. But today when this complication is commoner than ever, quinine is never used in antipyretic doses.

(16) *Age.* The age of the patients ranged from 16 to 55 years, but 71% of them were between the ages of 20 and 30 years.

(17) *Sex.* In the female wards eight cases of purulent otitis media developed in 106 cases of typhoid fever, (7.5%.) None of these cases developed a mastoid empyema. In the average the typhoid fever cases in the female wards were of a less degree of severity than those in the male wards; probably on account of the earlier taking the bed in the initial stages. As this simply brings us to the severity of the general infection, it cannot be said that sex is a factor.

(18) The bearing of nativity on the etiology is interesting. Of 89 otitis cases, the following were the nativities:

Austro-Hungary	35
Greece	1
Ireland	3
Italy	25
Poland	6
Russia (southern)	10
Russia (northern)	1
Slavonia	4
Syria	1
United States (negroes)	3

Of 30 mastoid cases, only two spoke English and those were of Irish birth.

The writers could not obtain the nativity of all the typhoid fever cases, but the United States was well represented, as almost every other large country, yet the purulent otitis seems to have occurred almost exclusively in the races of Southern Europe and Asia.

This preponderance of foreigners is due to the fact that for reasons before explained they have more severe typhoid fever infections, and this means more liability to purulent otitis, a more severe otitis, and a greater probability of mastoid empyema. The lack of cleanliness may also be a factor.

(19) Occupation records did not give data of any value for the reason that laborers so enormously preponderated that there were not enough of other occupations to figure percentages on. In the female wards, domestics preponderated, but there were not enough of even these to draw conclusions.

(20) Impacted cerumen was found in, and removed from, 18 ears out of 288 cases of typhoid fever examined. As only one of the 18 cases, and that only a slight impaction, developed otitis, no causal relation could be traced.

(21) Associated diseases of the nose and throat: These may be classed into typhoid and pre-typhoid, according as they developed during the typhoid fever or preceded it. As to the pre-typhoid 48 different pathologic states and deformities of the nose, naso-pharynx and pharynx were found as follows:

Adenoids	2
Naso-pharyngeal Bursitis (Tornwaldt's disease)	1
Tonsillitis, chronic, follicular	12
Tonsillitis, acute	1
Tonsil hypertrophied	18
Naso-pharyngitis, chronic	62
Naso-pharyngitis, ulcerative specific	2
Pharyngitis, chronic	62
Pharyngo-mycosis	1
Naso-pharyngeal neoplasm, fibroma (?)	1
Trachitis	26
Laryngitis, chronic	18
Laryngitis, ulcerative specific	2
Laryngeal paralysis, unilateral, abductor	1
Septum, deviation of	54
Septum, ulceration of, specific	1
Septum, spur of	22
Rhinitis, hypertrophic	71
Rhinitis, atrophic	2
Frontal Sinusitis, acute with empyema	2
Sinusitis, chronic	1
Ethmoiditis, chronic	5

As to the associated lesion of the nose and throat, that in all probability developed during the typhoid fever, the following were found in an examination of 280 cases:

Rhinitis purulent	13
Rhinitis catarrhal	72
Rhinitis ulcerative typhoid	6

Naso-pharyngitis	234
Pharyngitis	198
Thrachitis	52
Laryngitis	207
Tracheal perichondritis	16
Laryngeal "	8
Purpura of nasal mucosa	6
Purpura of naso-pharyngeal	2
Purpura of pharyngeal	3
Purpura of laryngeal.	
Septum, perichondritis of typhoid	2
Septum, perihondritis of specific	1
Granular swelling and localized soft hypertrophies in naso-pharynx	3
Anemia of nasal mucosa, with the dried secretion giving appearance of atrophic rhinitis	62

EXCITING FACTORS.

1. Extension by continuity of tissue may have occurred in the tubal cases before recited, though the writers doubt it. If it did not occur here, there was certainly no evidence of its having occurred in any other cases.

2. Infection by way of the external canal:

In the bacteriological examinations here recorded every canal proved sterile close to the drum membrane. Manifestly infection did not occur by this route. In cases before mentioned, the canal was found sterile a short time before the purulent otitis developed.

As a sample of hematogenous infection the case reported by Seidle is interesting. A typhoid fever case was complicated by mastoid empyema, axillary and temporal abcesses; all secondary to a liver abcess which developed on the 23rd day of typhoid fever.

We come now to consider the time-honored theory of ear infections, namely, infection by means of an infective mass passing up by way of the Eustachian tube, (not by extension of an infective process by continuity of tissue). We must not make the mistake of supposing that if the ear condition be a true typhoid fever process due to bacillus typhosis, that the bacillus necessarily reaches the ear per tubam. It has been found in tissues and organs remote from the alimentary or respiratory tracts, and not open to air borne infection. The conclusion is that it is carried by the blood, and there is no reason why it should not be so carried to the ear. Hewlett found 83 typhoid fever cases to have bacillus typhosis in the blood.

As to whether the middle ear conditions be an infection per tubam, the evidence seems to be that it is in the mild cases. As above recorded, pus was present in the naso-pharynx in all cases before the onset of suppuration. In every case examined after the onset of purulent middle ear inflammation, pus was found abundantly in the naso-pharynx. This was thick and ropy while the ear discharge (at that stage) was thin and bloody, so that the reverse order of things (tubal drainage of tympanic discharges) was excluded. We exclude here a number of cases where pus did not drain from the ear into the naso-pharynx. Tubal closure as a cause may be excluded because there were practically no closed tubes noted. In fact some tubes may have been too permeable, thus facilitating infection. Extension per tubam by continuity of tissue is probable. Extension by forcing bodily of a mass of infective material during sneezing, vomiting, coughing, swallowing, etc., is probable. Both of these probabilities are like the legal illustration of the value of circumstantial evidence: We see the puff of smoke from the revolver, hear the report, see the victim drop with a bullet hole in his body, and later, at the autopsy, the bullet is found. Nobody saw the bullet go. So it is with this. We certainly have the infective material at one end of the tube and an infective process later starts at the other end. It seems plausible that it gained access through the tube.

Dr. Thomas Arbuthnot, in searching for the source of infection erysipelatous and purulent, found the sterilization of bedding thoroughly defective, through the inefficiency and carelessness of a conscienceless employee, so that the pillows were at one time not really sterilized, simply recovered with clean linen cases. This seemed to account for three series of cases we had previously recorded. One bed had four consecutive purulent otitic complications in its typhoid fever occupants; another bed had two consecutive cases and the third bed had two consecutive cases, an intervening uncomplicated mild case of typhoid fever, and then another purulent otitis. In all these instances the first cases were fulminating, the later cases mild. This is a remarkable observation and points to the two types of purulent otitis, fulminating and mild being due to different causes, and very plausibly to embolism in the fulminating and infection in the mild cases. As to the route by which the infection reached the ear, it must have been by way of the nose and naso-pharynx, as the external canal was found sterile close to the drum membrane.

In one instance three cases of purulent otitis occurred in adjoining beds in one, two, three order, two and three days, respectively, apart. The means of infection could not be traced unless it were

from drinking out of each other's vessels, by turning over to the neighbor's table, which stood on the other side from the patient's own table.

As to the infection by way of the lymphatics and by the petro squasmosal fissure, we saw nothing on which to base a statement.

(3) Bezold reports finding in one case, post mortem evidences of extensive middle ear sloughing from the occlusion of the principal arterial blood channels of the middle ear by emboli.

The application of this hypothesis to the etiology of middle ear suppurations, harmonizes with all the observed phenomena, such as prompt rupture of the membrane, copious sanguinolent serous discharge becoming rapidly purulent in spite of efforts at asepsis and antisepsis, extremely sudden onset without previous catarrhal symptoms, etc., characterizing the cases of fulminating type.

Embolism of the auricular artery or its branch the stylo-mastoid or of the tympanic branch of the external maxillary or of the superficial petrosal would account for the fulminating cases. Anastomosis is very free, however, and perhaps in the adult the stylo-mastoid is the only one whose embolic occlusion would so deplete the tympanic mucosa as to render it necrotic or open to bacterial invasion. The chief defect we find in this theory is that the stylo-mastoid artery supplies a considerable extent of the neurilemma of the portio dura, yet no disturbance of the innervation of the muscles it supplies was noticed. Another point we would make is that the stylo-mastoid is not a "terminal artery" and thus not subject to infarct, as for instance the lungs and kidneys are. Still clinicians often see cases in which temporary or permanent low resistance added to impeded, but not occluded, circulation, will result in tissue necrosis.

Further, if we accept the embolic theory (a proven fact only in Bezold's solitary case) we must demonstrate an original source from which the emboli break away. This implies an endocarditis or an infective pus focus in the peripheral circulation. Endocarditis was rare, but pus foci were numerous, though they could not always be proven to exist prior to the ear affection. Frequently, however, they were found so shortly after the ear affection that it seemed justifiable to consider the remote pus focus primary, though not sufficiently manifest to be discovered until after the ear discharge, which was noted within a few minutes of its appearance by nurses by residents or nurses. We acknowledge the deficiency of our statistics in regard to the number of cases of endocarditis.

Pus foci remote from the ear and occurring prior to the ear trouble were found in six out of 38 (15.8%) mild ear cases and in 42 out of 50 (84%) fulminating cases. Most of these were in the lungs and

bronchi though a few occurred in locations where an embolus could not reach the ear without first passing through the lungs, a manifest impossibility, unless of bacterial size, followed by increase in size after passing. It seems plausible that the ear lodged embolus might be from an infective focus in the lungs this in turn arising from the enteric lesions; the lateness of onset point to this. It is to be regretted that autopsies could not be obtained in the few cases that died just in the incipiency of the fulminating otitis. Four deaths occurred at this stage, one from pneumonia, one from hemorrhage, two from perforation. It would have been interesting to search for infarcts elsewhere, as well as to examine locally. We trust this may yet be done by ourselves or some one else. While unable to bring forward post mortem evidence we believe that primary thrombosis, independent of embolism may be the exciting cause of the fulminating type of cases.

In other words, we consider it possible that the fulminating type of purulent otitis media cases, may have a pathology similar to that of leukophlegmasia.

One very stubborn argument that can be brought against every factor above enumerated is that purulent otitis media is more common now than ever. This would seem to require the finding of a cause operative now that did not exist before. The chief change has been in three things. Hydrotherapy is more extensively used, ventilation with draughts is more common, and there is a change in the nativity of the foreigners who fill our hospitals. They used to be Irish and Germans with a sprinkling of Scotch and English. Now they come chiefly from Asia and the South of Europe. This population is of excessively dirty personal habits, compared to our laboring class. It is remarkable, as before stated, that while the typhoid fever cases included every nationality, the purulent otitis cases occurred almost exclusively in natives of Southern Europe, and were the severe cases of typhoid fever.

SYMPTOMATOLOGY.

According to our observations there are three distinct types of purulent otitis media in typhoid fever, the hemorrhagic, the slow and the fulminating. In some cases of the hemorrhagic type, the blood filled the tympanum, causing pain until incised—other cases started as hemorrhagic blebs which were merged. Both types soon became purulent. The slow type does not differ materially from acute otitis as we usually see it, except in so far as it may be masked by the general malady. It is apt to be acute catarrhal at the onset, and become purulent later. Occasionally it seems purulent from incipiency.

The presence of pain depends upon the degree of toxemia. If the patient be profoundly toxemic, the discharge will be the first symptom, unless the ear has been watched daily. If the patient be not stupid from toxines, he will complain of pain for several days, when there will seem to be fluid in the tympanum, though the membrane will not be bulging much, if any. If not incised, it may not burst for a week after the onset. The appearance of the drum membrane does not differ from that of acute otitis. One difference is marked, however, namely, after the membrane is perforated by rupture or incision, the discharge will invariably become purulent in spite of the most earnest efforts at asepsis. The fulminating cases are of an entirely different type. The onset is exceedingly sudden. In ten cases no inflammatory phenomena were present 24 or 48 hours previous to spontaneous rupture of the membrana tympani. Upon examination, the drum membrane was found intensely red, a large perforation occupying most frequently the posterior inferior quadrant. Though it could be seen a swollen, deep red, hemorrhagic mucosa. Here was an intense otitis media purulenta fully established, with extensive pathologic change in an ear quite normal, or at least quite free from inflammation, in some instances 24, in others 48 hours before. Possibly, the time was even shorter, but it was examined by one of us at about those intervals. The membrane had already ruptured at the end of this short time, if not, of course, we incised it. Then a very abundant sero-sanguinolent discharge commenced to pour out of the meatus to the extent of three or four ounces daily. If the patient would turn his head to one side, the upper ear would fill with this discharge all the way to the concha, which would also fill, in sometimes, half an hour. When he would turn, he would dump this concha full of discharge onto the pillow. All three types of purulent otitis, the hemorrhagic, the slow and the fulminating were accompanied by more or less deafness and tinnitus, but these were often equally present in cases that had not middle ear inflammation.

Sensations of fullness and other subjective signs of closure of the tube by tumified membrane were sometimes present after the onset of the otitis. Many cases of both types also had a tenderness during the early stages, located in the soft tissues over the mastoid. This always subsided in a few days. Usually the patient had not noticed the early tinnitus or the early impairment of hearing. In the slow cases, pain was occasionally complained of by the patient at the onset, but if an otoscopic examination had been made every day it was noticed that the pain was preceded some hours by a congestion of the membrana tympani, first along the manubrium, later

spreading to the periphery. In the fulminating cases there was often pain, and when present, it was severe at the outset, but subsided completely in a few hours. If the toxemia was great, pain was not complained of. In the fulminating class, the drum membrane, in many cases did not get a very intense red, for soon an exfoliation from its outer surface began, which gave it rather a grayish appearance, sometimes dark sometimes nearly white. Exfoliation from the deeper canal also often occurred. The inflammation became more intense, the canal and the mastoid became tender, the lymphatics near the ear sometimes swelled (though this usually occurred later) the membrane began to bulge and if not incised, burst usually in the posterior inferior quadrant. The upper and the posterior boundaries of the membrane would disappear so that the membrane would seem continuous with the canal walls in these regions. With the beginning of the discharge the maceration and exfoliation of the epithelial layer of the drum membrane and of the deeper canal wall would increase until it nearly filled the canal. Inspection would be impossible until after a thorough mopping of the parts. The discharge was serous, or, more frequently, sero-purulent, almost invariably sanguinolent, in the early stages; later becoming thick, creamy pus of a dirty, pale greenish color and without fetor unless there had been pre-typhoid chronic purulent otitis. In one case without previous ear disease it had a fecal odor.

The discharge in 22 cases out of 88 was purulent from the beginning though the pus was diluted with serum and mucus. In 66 cases it was serum tinged with blood and very copious in quantity, in some instances reaching probably two or three ounces daily. The quantity of dischage usually reached its maximum with a week by which time, it was thick, creamy pus. Then the quantity usually began to diminish, and if the case were one that tended toward chronic otorrhea the discharge persisted in a very much diminished quantity. In the cases that later developed mastoid empyema, the discharge also grew less in quantity and persisted at the diminished rate and sometimes ceased altogether, due to obstruction.

Tenderness on Pressure over the mastoid was present in 23 of the 88 otitis cases in the first few days of the otitis. At first it was thought this was due to a periostitis from extension of the infection out behind the fibrous canal where we often see pus find its way in children. But the quick subsidence of this tenderness and the absence of swelling precluded periostitis.

The Temperature in both types if it had reached normal, would usually rise with the development of the otitis and subside with the rupture or incision of the drum membrane. Even in the cases of

mastoid empyema it usually remained normal or nearly so, except in one case, where operation had been postponed for special reasons, until the pus burst through into the soft tissues of the neck.

Otorrhagia, in eight cases hemorrhagic blebs were noted on the external surface of the drum membrane, the accumulation of sanguinolent serum (sometimes almost blood alone) was evidently under the outer epithelial layer of the drum membrane, as the blebs readily burst when touched with a probe. In several of the cases the blebs extended beyond the periphery of the membrane raising the epithelial layer of the canal. In every case where the blebs were noted, a purulent middle ear inflammation developed later. Probably inflammation existed at the time, but pus formation came later. In two cases the middle ear, when evacuated by incising the bulging drum membrane, was found to be filled with the same fluid as the blebs. In one of them the fluid was also seen in the Eustachian orifice in the naso-pharynx. In one case of purpuric typhoid the eruption was seen upon the tympanic mucosa by inspection through a perforation enlarged by incision. It is not common to see the early discharges of an acute otitis media blood stained, but in these cases it is the rule. It is probably an oozing from a highly inflamed mucosa. In one case a genuine hemorrhage from the ear occurred. The patient, a man 29 years old, had an intestinal hemorrhage two days previously. Thick, dark blood was noticed dropping from the auricle to the pillow. It was not mixed with pus or catarrhal discharge, but seemed to be blood alone. Upon wiping the canal out a perforation was noticed in the membrane flaccida, through which the blood was pushing in a small bead, increasing in size until a drop was formed, and continuing to flow outward. The aural hemorrhage continued for two days as blood alone, then began to gradually change to bloody pus, later to the usually grayish greenish, yellow pus unmixed with blood. As to the source of this hemorrhage it certainly came from the middle ear, as a probe freely entered the perforation. Whether it came from a vessel eroded by a typhoid ulcer or not can only be conjectured. Yet, as it was impeded and followed by a typical intestinal hemorrhage there might be grounds for believing that it was similar pathologically. It did not, as in the purpuric cases, seem to lack the clotting quality. It is to be regretted that the interior of the middle ear was not examined in this case by free incision of the membrane. In another case, however, where an incision of the drum membrane was necessary to allow of free drainage in a bulging unperforated membrana tympani, occasion was taken to encircle the entire drum membrane and elevate it as a flap to thoroughly examine the middle ear. An

ulcer was discovered on the promontory, quite superficial. Later it became deeper and the membrane became more swollen and œdematous, until all landmarks were obliterated. At the end of three weeks, however, the discharge had ceased, the drum membrane had united all around the circular incision and the ear was practically well, though the hearing did not quite reach the normal. The linear shape of the ulcer was suggestive of thrombosis of a small vessel.

Deafness, to a greater or less degree was always present in the affected ears, but as it was always present in the unaffected ears to an almost equal extent, it was probable that the perceptive apparatus, including the sensorium, was not in condition to perceive delicate sounds, even if the conducting apparatus would transmit them. The hearing power was not easy to determine for many reasons. Hebetude, stupidity, delirium, unconsciousness, lack of an interpreter; these, and other things, narrowed down the hearing tests to 98 cases of typhoid fever. Of these, 48 (49%) could not hear a watch tick. Fifty-four (55.1%) could not hear a low voice at two feet.

Tinnitus was rather difficult to get statistics on for the reason given under deafness. In 20 cases of purulent otitis media that we were certain understood our meaning, 17 (85%) had tinnitus. Tubal symptoms, about which it would seem interesting to gather statistics were impossible of comprehension by our interpreters, to say nothing of getting it into the patient's disordered sensorium.

DIAGNOSIS.

The diagnosis of otitis media in typhoid fever, does not differ greatly from that of the same disease in other infections. Provided the ear is examined every day; but if one waits for the patient to complain of the pain or tinnitus, the diagnosis will be made by seeing a copious ear discharge in about 50% of the cases. This refers to hospital cases.

The diagnosis between a primary acute purulent otitis media that has not been incised and drained, has become sub-acute, has involved the mastoid, and a typhoid fever with ear suppuration may not seem difficult, and is not, to the otologist, but the general practitioner will often experience difficulty, especially where clear histories cannot be obtained. Such a case complicated with pus foci elsewhere, where absorption is freer than in the ear and mastoid, may have a temperature chart, simulating typhoid fever closely. The Widal reaction will be of assistance.

PROGNOSIS.

As to Life, of 88 cases of purulent otitis media in typhoid fever none were fatal through any complication of the ear condition. Four died but of conditions remote from the ear; one of intestinal perforation, two of toxemia, one of intestinal hemorrhage. That death did not occur from the ear complications was due solely to the fact that all cases were watched, that free drainage through an ample perforation or incision was maintained, and that the mastoid involvements were cut short early. If free drainage be not maintained, erosion through the tympanic roof or elsewhere may put the patient's life in jeopardy. If, in the mastoid cases, operation be postponed too long, a large element of risk is introduced. In no other class of mastoid cases do we see more rapid tissue destruction. In very pneumatic mastoids the cell walls seemed to have almost melted away. Hence the prognosis is favorable, only in early operated cases. Of the otitis media purulenta cases, 31 (35.2%) recovered without a perforation. Twenty-six (29.5%) recovered with a perforation; sixteen (18.2%) were discharged with *otorrhea* because they refused operation, and in 15 (17%) the condition on discharge was not recorded. There was suppurative *mastoid involvement* in 26 cases out of the 88 (29%). *Tinnitus* was still present in about six out of 26 (23.1%) cases when discharged. It was especially bad in one case in which a radical operation had to be done. This case had a previous chronic otorrhea. Possibly he had tinnitus prior to his typhoid, though he denied it. *Deafness* was total and permanent in two cases, fortunately both unilateral. These two were suppurative involvement of the labyrinth secondary to the otitis media, really "panotitis.' Excepting these two, no case of purulent otitis media, including the mastoid cases, were unable to hear low voices at two feet, and only four were unable to hear a loud ticking watch on contact. This means four out of 32 (12.5%) for on account of previous ear disease, independent of labyrinthine trouble, or inability to get a record of hearing on admission, many had to be excluded. The hearing was better in the early operated mastoid cases than in the later operated cases, and was better in the early operated cases than in the suppurative otitis cases that recovered without mastoid involvement.

Impairment of hearing was, in ten cases, associated with lowering of the upper tone limit and these were excluded. The duration of the otorrhea in the cases that recovered before leaving the hospital and without the mastoid operation, was three weeks in the shortest case, eighteen weeks in the longest. A complete cure resulted in all the cases in which the mastoid was opened. This included two cases

with pre-typhoid chronic purulent otitis, in which a radical operation was done. It would be interesting to know just how many of the cases discharged with otorrhea who refused operation became chronic. Doubtless most of them did, though given instructions for the use of the boric acid douche, in their condition of life, it is probable they never carried out the instructions. The case books, both private and dispensary, of most otologists, contain few histories of ear trouble referred back to typhoid fever. Moos reports two, Bezold, 5; of the latter, two still discharged, one had a healed and a discharging ear, one case had normal ears; two of them showed darkened drum heads. One remembered an otorrhea ceasing 15 weeks after his typhoid fever.

TREATMENT.

Treatment would naturally divide itself into prophylactic and curative. The prevention of cases of fulminating type, if our opinion as to the thrombotic or embolic etiology be correct, we fear is beyond the skill of modern medicine. The prevention of the cases deemed infective would seem equally hopeless, if the process be due to blood-borne pyogenic organism or typhoid bacilli. If the purulent otitis be deemed primarily a direct infection by pyogenic bacteria, from the throat, there would seem to be ample room for prophylactic effort. Our investigations prove that infection probably enters not by the exernal canal, but by way of the tube. If this is the case the greatest efforts should be made to keep the air as free as possible from pyogenic organisms. All bedding, including mattresses and pillows should be fumigated as thoroughly after each patient (pus case or not) as after a case of smallpox.

Fumigation of the wards during which the patients are removed should be frequently done. The walls should be washed down with an antiseptic solution or painted, and the floors scraped and varnished. These and many other well known details ought to help. All draughts should be avoided, though thorough ventilation must be maintained. The head should not be too low, as this would favor a flow to the mastoid antrum, thus interfering with the drainage and possibly favoring hypostasis in the tympanic mucosa. Demeurisse urges isolation in all purulent otitis cases, especially those complicating the acute infections. Bezold recommends removing stagnant secretions by swabbing the naso-pharynx with an antiseptic solution, followed by insufflation of boric acid. Locally, antiseptic agents, but more still, cleansing agents, must certainly be used in clearing the naso-pharynx and tube mouths. The nose had probably better be left alone. A post nasal douche would be more than usually dan-

gerous. In all the prophylactic measures, one thing must be clearly borne in mind, namely, anything that excites or annoys the patient is a detriment to his general condition, so that we must be very sure that what we do is of unquestioned value. For this reason, Politzer's method of douching through the tube, by means of a catheter for cleansing suppurative ears, was not used after a single trial. It is a good measure, but was so fought against and dreamed and fretted about by the patient, that it was abandoned. The regular toilet of the mouth is agreeable to the patient and is certainly a good thing from the otologist's as well as the attending physician's viewpoint.

The ears of every typhoid fever patient after the second week should be examined, cerumen removed if present, and a daily watch kept on the drum membrane. When its vessels along the manubrium become visible, start at once the hot douche, antiseptic if you like, but hot. When the drum membrane reddens, it should be incised under aseptic precautions, without delay. If it prove but a myringitis, no harm will have been done, and usually it will be found to be the early stages of purulent median otitis.

The advantage of free drainage was shown in the six cases where the membrana tympani was circumcised and in five cases quartered. This though done for inspection, by the free drainage afforded evidently was a most efficient therapeutic measure, for the cases not only recovered from the otitis, but recovered without leaving a perforation. A fear that in the low recuperative power of typhoid the membrane might slough proved groundless. It would seem that occasionally one ought to succeed in preventing infection of the discharge when not already purulent, but our efforts to prevent purulency were utterly unavailing for the reason that the intricate recesses of the tympanum were already affected as mentioned under *"bacteriology."* Pus there was not, in many cases, but a few pus cells were visible microscopically, and worst of all, invariably pyogenic organisms. After the incision the canal should always have a wick of iodoform gauze to facilitate the drainage. The gauze should be removed every hour or two and a thorough douching given and then it should be replaced.

Frequent douching is essential to keep the canal clean, to prevent pus from thickening on the canal walls, to prevent plugging of the perforation. That the antiseptic had no effect was clearly proven by the equal results from the use of salt solution. The heat and moisture probably had some inhibiting influence on the inflammation. Be the modus operandi what it may, certain it is, that mastoid

empyema was much less frequent after systematic douching was instituted, though figures are not available.

In the early stages of otitis media in typhoid fever, the discharge was so profuse that all air infection is washed away in the flow; but as soon as the flow checks, air infection occurs. With a view of preventing this, the canals of a series of 20 cases were treated as a septic wound, packing with iodoform gauze immediately after an aseptic incision in the drum membrane. These cases became purulent just as soon as in a series of 20 cases treated simply by frequent douching showing that the purulency comes from within. The chemicals in the douche make little if any difference. We first used normal salt solution so as not to interfere with bacteriologic culture tests, but we soon found it quite as effective as any active germicide. The main thing is heat. The temperature should not be less than 110, and the douching must be frequent.

COMPLICATIONS.

The only complications were erysipelas and mastoid empyema. Erysipelas occurred in three cases. It was located on the face and scalp of all, but it seemed doubtful whether the infection was secondary to the ear condition. It occurred in a number of cases of typhoid fever uncomplicated by otitis, or any other lesion. It did not seem to influence the otitis in any way. Purulent otitis seemed to result from extension of erysipelas in one case.

Mastoid empyema is certainly growing more frequent as a complication of otitis in typhoid fever. Hengst found but one case of mastoiditis in 20 suppurative otitis cases complicating typhoid fever. Bezold found but one in 43 cases. But the older authorities never seem to have found mastoid empyema in purulent otitis from any cause as frequently as it is found today. Mastoid empyema occurred in 26 out of 88 (29.5%) of the purulent otitis media cases. Mastoid tenderness occurred without pus formation in 31% of the cases. This appeared at the outset of the malady, and always disappeared, in some cases not re-appearing, and in others returning with the pus formation in the mastoid cells. The large percentage of mastoid complications in the purulent otitis cases, is due probably to three causes, virulence of the infection, lowered resistance, and the almost constant dorsal decubitis which favors the collection of pus in the antrum. This is specially true of cases where on account of intestinal hemorrhage or other reason, the pillow is removed and the foot of the bed is elevated. Gravity has no opposition on account of the absence of cilia in the mucosa of the aditus ad antrum.

It is a remarkable observation that every purulent otitis case so treated (by elevation of the foot of the bed) that did not die too soon (of conditions remote from the ear) developed a mastoid empyema. There were four of these cases, one unilateral, and three bilateral. All the bilateral cases developed bilateral mastoid empyemata. The low resistance was shown on one case, where, after cleaning out a mastoid down to healthy bone, the first dressing revealed large patches of bone, dead because it had not sufficient vitality to resist invasion. There were no peculiar features about the mastoid cases except the exceedingly rapid destruction of tissue and the fact that the temperature was absolutely normal for a week or more, before the operation, in 14 out of 26 (53.8%) cases. Evidently there is little power of absorption in the mastoid portion of the temporal bone. When the pus breaks through into the soft tissues of the neck, as it was allowed to do in one case, the usual pus chart appeared. In six cases out of 26 (23.1%) the temperature ranged to 99 or 100 and back to normal. In five out of 26 (19.2%) the range was to 104 and back to 90 or 100, but in these cases we could not exclude the possibility of its being due to pus in other locations or to a typhoid fever reinfection. The authors hope that it will not be considered boastful when the statement is made that of the 26 cases in which the mastoid was opened, pus was found in every instance; not only in the antrum but in the mastoid cells. This required careful watching. Had there been any doubt in our minds as to the presence of pus in the mastoid, in any case, we should certainly have operated anyway.

But to the otologist who watches a case every day, there is no difficulty in saying when pus begins to form. All save one were opened before pus had bursted through the cortex. In this one case operation had been postponed for a special reason for two weeks beyond the time when operation was deemed advisable.

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